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CASE REPORT

A unique iatrogenic organized left atrial tachycardia with a gap conduction in previously ablated lesions

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Summary This case concerns a 57-year-old woman with an organized left atrial tachycardia (AT) after pulmonary vein (PV) isolation. The left inferior PV (LIPV) exhibited a figure of eight tachycardia around the LIPV ostium with one loop at the anterior aspect and another at the posterior aspect, which corresponded to regular surface P waves. Although a gap ablation of fractionated electrograms changed the LIPV sequence, the atrial potentials were organized with a shortened cycle length accompanied by the same surface P-wave morphology. The elimination of the anterior loop and modification of the posterior loop after the gap ablation might have yielded that specific sequence change of the AT.

Introduction

Atrial fibrillation (AF) is related to cardiovascular events and symptomatic arrhythmias [1,2]. Electrical isolation of the pulmonary veins (PVs) to treat drug refractory AF is becoming more common. That has led to the development of PV isolation in patients with AF utilizing either small segmental lesions guided by a circular catheter [3,4] or large continuous circular lesions some distance from the ostia of the PVs [5]. However, such lesions may lead to iatrogenic left atrial (LA) macroreentrant atrial tachycardia (AT) [6—8]. The mechanism of this arrhythmia is controversial and difficult to establish. We report a unique iatrogenic organized LA tachycardia with a gap conduction in previously ablated lesions.

Case report

A-57-year old woman was referred for treatment of paroxysmal AF. Her arrhythmia was refractory to antiarrhythmic drugs including pilsicainide, flecainide, and bepridil. Her history revealed no evidence of cardiovascular disease, and her physical examination and X-ray revealed no abnormal findings. Echocardiography revealed no structural abnormal-

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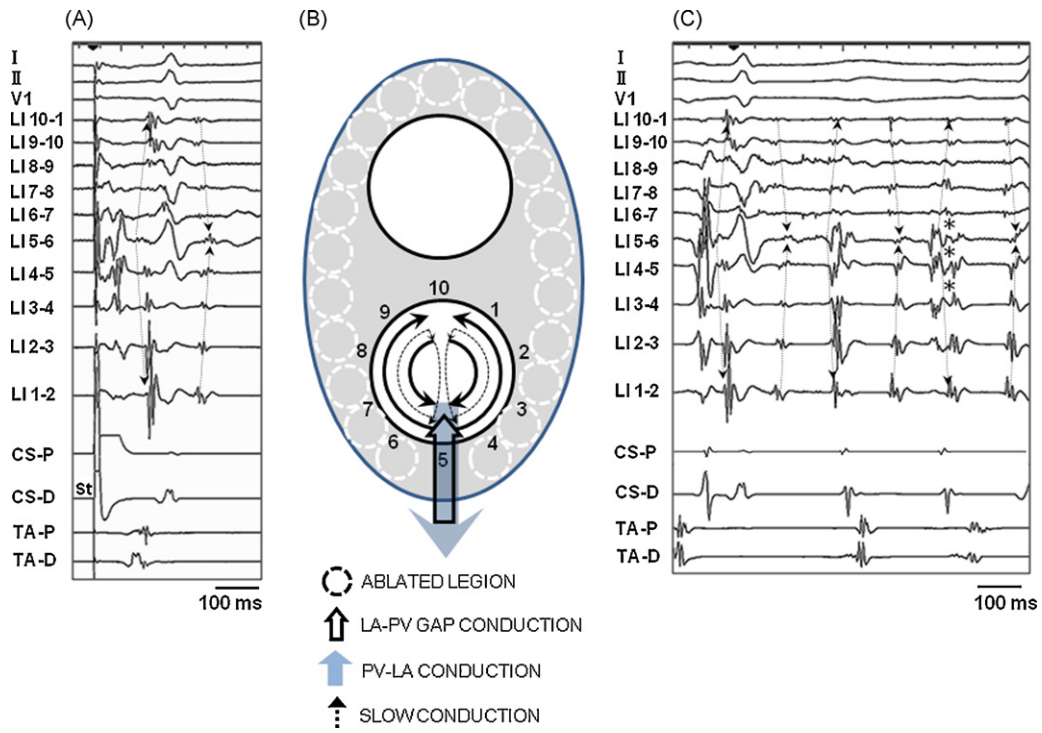

Figure 1 Tracings of ECG leads I, II, and V1 and the intracardiac electrograms recorded from the coronary sinus (CS) catheter and a circular catheter located in the ostium of the left inferior pulmonary vein (LIPV). (A) CS pacing induced a specific activation pattern in the LIPV. (B) Schematic representation of the activation pattern at the left PV ostium. The conduction might have occurred via a figure of eight tachycardia with one loop at the anterior aspect (LI-5 to LI-10) and another at the posterior aspect (LI-10 to LI-5). (C) Initiation of the clinical atrial tachycardia (AT). Note that fractionated electrograms, reflecting slow conduction due to a gap conduction, were recorded around the exit site (asterisks) of the AT. LI1-10, the distal to proximal electrode pairs of the LIPV circular catheter; CS (TA)-D, CS (TA)-P, distal and proximal electrode pairs of the CS (TA; tricuspid annulus) catheter; St, stimulation.

ities (left atrial diameter: 36 mm, ejection fraction: 65%, long-axis view). She underwent extensive ipsilateral PV isolation. Radiofrequency (RF) energy was applied at a distance from the PV ostia guided by Lasso catheters placed within the ipsilateral superior and inferior PVs [3]. An encircling ablation line was created approximately 0.5—1 cm from the angiographically and electrophysiologically defined PV ostia without electroanatomical mapping, and all four PVs were successfully isolated [9]. Before and after RF application, no spontaneous AF triggers were identified with isoproterenol (ISP) administration (12 μg/min). Two days later she suffered from palpitations and her 12-lead electrocardiogram (ECG) revealed an AT with an isoelectric interval between regular P waves. At baseline, the AT persisted, and the ECG exhibited biphasic P waves in the inferior leads and positive P waves in V1 and V2. The AT could be terminated transiently by antiarrhythmic drug provocation (pilsicainide) or cardioversion, but recurred promptly. After obtaining written informed consent, an electrophysiologic study was re-performed. During sinus rhythm, isolation of the ipsilateral right PVs and left superior PV, and reconnection of the left inferior PV (LIPV) to the left atrium (LA) was confirmed. A specific conduction pattern around the LIPV ostium emerged during coronary sinus (CS) pacing (Fig. 1A and B). That activation sequence was not observed in the previous session. The clinical AT spontaneously emerged without an ISP infusion (Fig. 1C) or was easily induced by CS pacing reproducibility. During the AT, the LIPV exhibited an atrial activation sequence with a cycle length of 290 ms, which corresponded to the surface P-wave interval. The rest of the atrial potentials were all similarly organized (Fig. 2 left and lower left middle panels). Guided by a single circular catheter (Lasso, Biosense-Webster, Inc., Diamond Bar, CA, USA), a conduction gap was identified at a previously ablated lesion on the posterior aspect of the left inferior PV. Entrainment pacing at that site (bipolar pacing with 5 mA and a 2 ms pulse width) could not capture the PV or LA. A RF application using a conventional 4-mm-tip ablation catheter (Ablaze, Japan Lifeline, Tokyo, Japan) at that site exhibited continuous activation from the PV to the LA and transiently terminated the AT, and the intra PV tachycardia cycle length shortened (Fig. 2 lower right middle panel). During that tachycardia, the cycle lengths of the CS potentials shortened (290—270 ms) and also became organized with the same P-wave morphology as the recorded ECG (Fig. 2 right panel). An additional RF ablation application at that site completely terminated the AT, while the PV activation persisted within the left inferior PV (Fig. 3). After the last RF application, disconnection of the LIPV from the LA was confirmed and no atrial tachyarrhythmias could be induced by burst atrial pacing with an ISP infusion. Twenty months after the procedure, the patient has had no recurrence of the tachyarrhythmia without the use of any antiarrhythmic drugs.

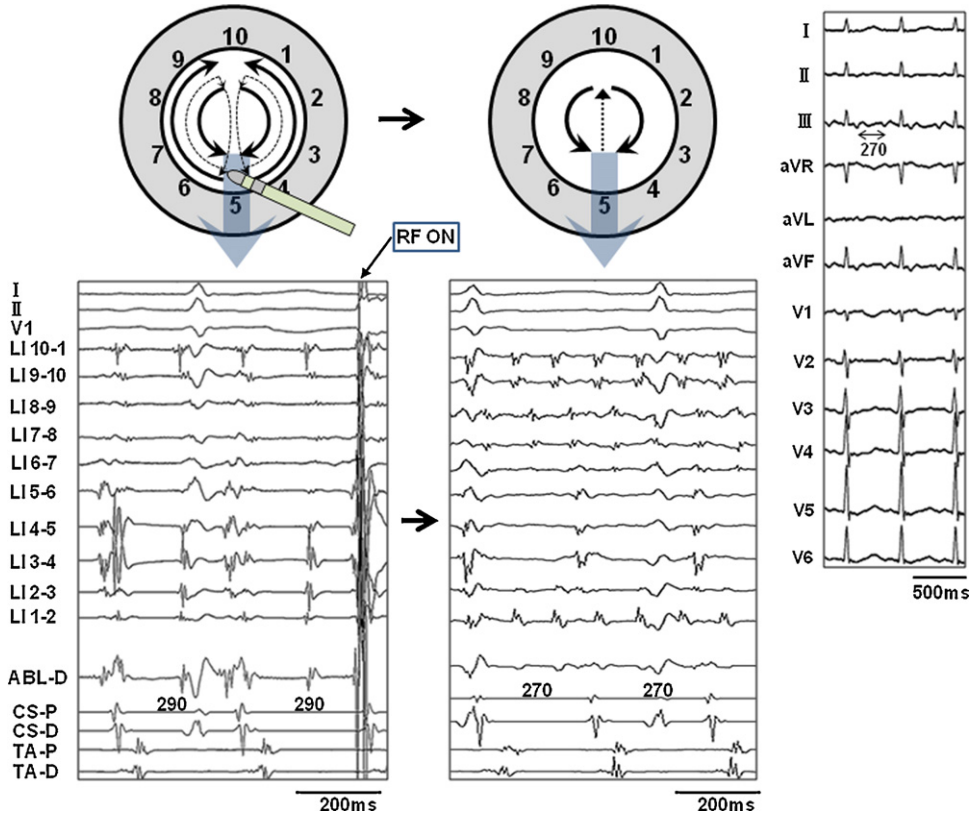

Figure 2 Intracardiac electrograms during the atrial tachycardia (AT) (left lower panel) and schematic representation of the estimation of the reentrant circuit (left upper panel). The AT cycle length became shortened by the first radiofrequency (RF) application (middle lower panel), suggesting the elimination of the anterior loop and modification of the posterior loop just after the gap ablation (middle upper panel). It is noteworthy that the coronary sinus (CS) potentials shortened (from 290 ms to 270 ms) and also became organized and exhibited the exact same P-wave morphology as the recorded ECG (right panel). ABL-D, distal electrode pairs of the ablation catheter. The abbreviations are as shown in Fig. 1.

Discussion

In this case, a unique LIPV activation pattern was documented during both CS pacing and the clinical AT. Although accurate positioning of the circular catheter around the LIPV ostium was difficult, the circular catheter revealed that one activation wavefront traveled around the anterior aspect (from LI-5 to LI-10) and another around the posterior aspect (from LI-10 to LI-5). Fractionated electrograms, presumably reflecting a slow conduction isthmus due to a gap conduction [8], were also recorded around the exit site of the AT (Fig. 1C). These fractionated electrograms may indicate that the anterior aspect of the wavefront is located outside the ablation line, while the posterior aspect of the wavefront is located inside the ablation line. Since a basket catheter was not inserted for detailed mapping inside the PV, and a detailed entrainment mapping study could not be obtained, it was difficult to reveal the mechanism of these arrhythmia events. One plausible explanation of the LIPV ostium activation was that the conduction might have occurred via a figure of eight type reentrant tachycardia with one loop at the anterior aspect and another at the posterior aspect (Fig. 1B). The reason that no fusion was demonstrated during the entrainment pacing maneuver at the LIPV ostium might be that the circuit of the AT was smaller than that previously reported [6,8,10].

During the RF application at the exit site with fractionated electrograms, the AT suddenly changed to a shorter cycle length with the exact same P-wave morphology. This finding suggested that the ablation site might have been a portion of the critical isthmus of the AT. Elimination of the anterior loop and modification of the posterior loop just after the gap ablation might have caused this specific sequence change of the AT (Fig. 2).

Limitations

Focal automaticity provoked by RF energy was also suspected as the mechanism for this event because the AT cycle length shortened just after the RF application. Furthermore, that substrate might have been created during the first session because those peculiar conduction properties of the LIPV were not observed in the previous session. Although the mechanism of the AT was unclear, our observations support the hypothesis of an interaction between the LA and LIPV via a gap conduction through a previously ablated lesion, contributing to the perpetuation of those organized left ATs using the same exit site to the rest of the atrium. To the best of our knowledge, there have been no reports of an AT following AF ablation occurring in this manner. A unique property of man-made gap conduction may have been the causative factor of this event.

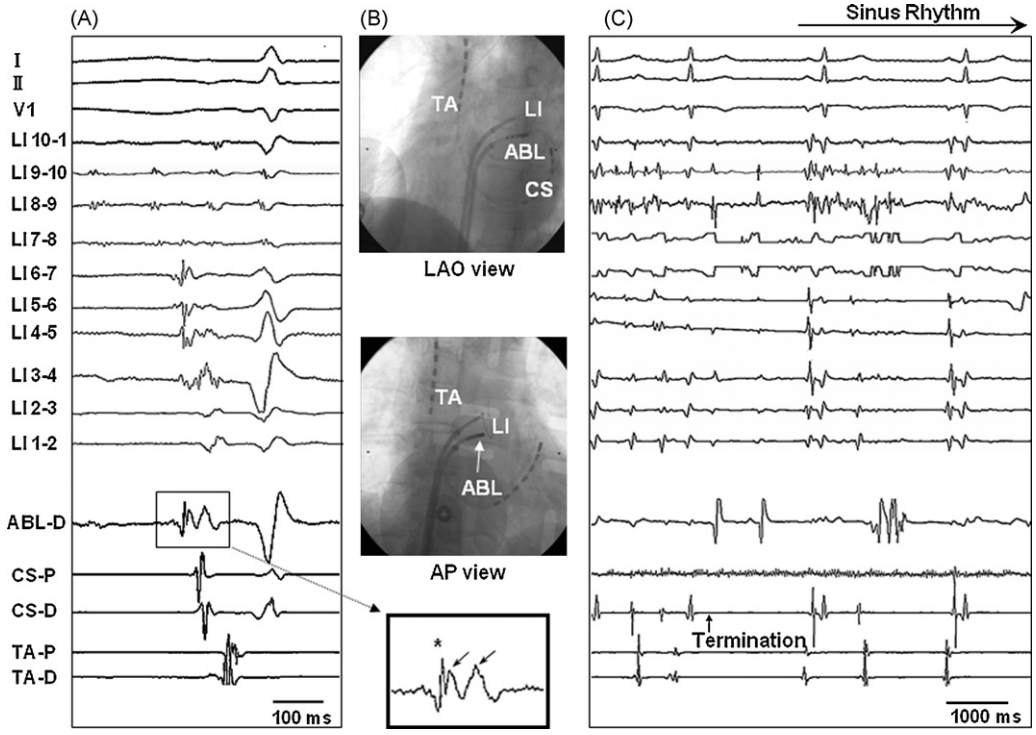

Figure 3 (A) Intracardiac recordings demonstrating an atrial tachycardia (AT) after the first radiofrequency (RF) application and the successful ablation site that terminated the AT. The electrogram recorded from the distal electrodes of the ablation catheter exhibited a continuous pulmonary vein (PV)-left atrial (LA) activation. The asterisk indicates the PV potential and the arrows may indicate the LA potentials. (B) Left anterior oblique (LAO) and anterior—posterior (AP) views of the ablation sites. The ablation catheter (ABL) was located at the posterior wall of the LIPV. (C) The AT was completely terminated during the second RF application, while the PV activity persisted in the left inferior PV. The abbreviations are as shown in Fig. 1.

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